Amendments to the Specification:

Please amend the specification as follows:

Please amend the title of the invention as follows:

Combination Therapy For Treatment Of A Disorder Characterized By Cells Expressing The CD40 Surface Antigen

Please insert the following sentence after page 1, line 1:

This application claims priority to USSN 60/280,805, filed April 2, 2001.

Please replace the paragraph beginning at page 20, line 3, with the following amended paragraph:

A preferred agent within the context of the present invention is an agent based upon the binding determinants of the monoclonal antibody S2C6 (Paulie et al., (1984) Cancer Immunol. Immunother. 17:165-179). S2C6 comprises the V_L amino acid sequences of SEQ ID NO:1 and SEQ ID NO:2 and the V_H amino acid sequences of SEQ ID NO:6 and SEQ ID NO:7 of WO 00/75348. While S2C6 has been shown to have an agonist activity on human peripheral B cells as demonstrated by its ability to stimulate primary B cell proliferation in a dose dependent manner (Paulie et al., (1989) J. Immunol. 142:590-595), agents based upon the antibody have been shown to have anti-neoplastic activity in vivo (International Publication No. WO 00/75348).

Please replace the following paragraph beginning at page 21, line 30, with the following amended paragraph:

The agents of the present invention directed against the CD40 and the CD20 surface antigens ~~may~~ may be administered in combination with other growth arresting agents. Examples of growth inhibitory agents that do not bind the CD40 or CD20 membrane antigen include agents that block cell cycle progression (at a place other than S phase), such as agents that induce G1

arrest and M-phase arrest. Classical M-phase blockers include the vincas (vincristine and vinblastine), TAXOL®, and topo II inhibitors such as doxorubicin, epirubicin, daunorubicin, etoposide, and bleomycin. Those agents that arrest G1 also spill over into S-phase arrest, for example, DNA alkylating agents such as tamoxifen, prednisone, dacarbazine, mechlorethamine, cisplatin, methotrexate, 5-fluorouracil, and ara-C. Further information can be found in The Molecular Basis of Cancer, Mendelsohn and Israel, eds., Chapter 1, entitled "Cell cycle regulation, oncogenes, and antineoplastic drugs" by Murakami et al. (W B Saunders: Philadelphia, 1995), especially p. 13.

Please replace the following paragraph beginning at page 22, line 8, with the following amended paragraph:

In a preferred embodiment the agents which ~~arrests~~ arrest the growth of, ~~destroys~~, destroy or ~~causes~~ cause the deletion of cells expressing CD40 and CD20 are antibodies. A description follows as to exemplary techniques for the production of the preferred antibodies used in accordance with the present invention.

Please replace the following paragraph beginning at page 45, line 2 with the following amended paragraph:

Ramos EBV-negative Burkitt's lymphoma, HS Sultan EBV-positive plasmacytoma and IM9 EBV-positive multiple myeloma cell lines were purchased from American Type Culture Collection (Manassas, Va. 20110). Rituxan® resistant Ramos lymphoma cell line was established through exposing the Ramos lymphoma cell line to high doses of Rituxan® (500 ug/mouse IP, 3 times/week for 3 weeks) in a subcutaneous xenograft scid mouse.

Please replace the following paragraph beginning at page 45, line 10 with the following amended paragraph:

Mice were injected through the tail vein with 1x10e6 tumor cells in 100 ul HBSS. Treatment with control antibody or SGN-14 or Rituxan® or SGN-14 and Rituxan® combination or chimeric SGN-14 mAb or chimeric SGN-14 mAb and Rituxan® or human anti-CD40 mAb was started on day 3 after tumor inoculation. The antibodies were given intraperitoneally 100 ug per mouse in 100 ul sterile saline at the frequency of 3 times a week for a total of 3 weeks treatment. The mice were monitored twice daily for mortality. The cause of death was confirmed by histopathological evaluation.

Please replace the following paragraph beginning at page 45, line 20 with the following amended paragraph:

Mice were injected subcutaneously at the right flank with 5x10e6 tumor cells in 100 ul HBSS. Treatment began when the tumor volume reached about 150 cubic mm with either control antibody or SGN-14 or Rituxan® or SGN-14 and Rituxan® combination or chimeric SGN-14 mAb or chimeric SGN-14 mAb and Rituxan® or human anti-CD40 mAb. Each mouse received 100 ug of one of the antibody in 100 ul sterile saline intraperitoneally 3 times a week for a total of 3 weeks treatment. The tumor volume was measured weekly.